

1919
M66

Miner - Experimental survey of Guinea pig. 1919.

**EXPERIMENTAL SCURVY
OF THE GUINEA PIG
THE ANTISCORBUTIC VALUE OF MILK
AND MILK PRODUCTS**

BY

HELEN NELLORA MINER

THESIS

FOR THE

DEGREE OF BACHELOR OF SCIENCE

IN

GENERAL SCIENCE

COLLEGE OF LIBERAL ARTS AND SCIENCES

UNIVERSITY OF ILLINOIS

1919

1919
1900

UNIVERSITY OF ILLINOIS

June 14, 1919 190

THIS IS TO CERTIFY THAT THE THESIS PREPARED UNDER MY SUPERVISION BY

Helen Nellora Miner

ENTITLED Experimental Scurvy of the Guinea Pig. The Antiscorbutic Value
of Milk and Milk Products.

IS APPROVED BY ME AS FULFILLING THIS PART OF THE REQUIREMENTS FOR THE

DEGREE OF Bachelor of Science

General Science

Howard B. Lewis

Instructor in Charge

APPROVED:

W. A. Hayes

HEAD OF DEPARTMENT OF Chemistry

443958



TABLE OF CONTENTS

Resume of the Disease of Scurvy

Experimental Scurvy

Conflicts between various workers

Problem

Diagnosis of Experimental Scurvy

Experimental Work

Series I

Series II


Tables

Charts

Conclusions

Acknowledgment

Bibliography



Digitized by the Internet Archive
in 2013

<http://archive.org/details/experimentalscur00mine>

Scurvy is by no means a new disease and altho recent work has brought it before the eye of the scientific worker we have record of its appearance as far back as the time of the Crusaders. In de Joinville's account of the Crusade of Louis XI we are told that scurvy attacked the troops in Palestine. This malady was very common among the crews of the old sailing vessels which were often at sea for a period of many months and were frequently deprived of fresh foods. We have a record of a scurvy epidemic in Dublin in 1847 and if we turn to the various military records we find that scurvy was at one time the scourge of the army. In the Medical and Surgical History of the War of the Rebellion during the five and one-half years covered by the statistics there are reported 30,714 cases of scurvy among the white troops with 383 deaths attributed directly to that disease. This report states further that scurvy developed at practically all of the military posts during the winter seasons. At posts which could be readily supplied with potatoes there was only a slight trace of the disease. But of still greater interest are the reports of the appearance of scurvy during the recent World War. It has not only affected the troops but the civilian population as well. Harvier (1917) a French surgeon states that 95 percent of the 800 troops of which he had charge suffered from scurvy and later other epidemics centers were recognized outside this sector. Another author reports scurvy among the Italian troops. Germany likewise suffered from this disease if we can judge from the condition of the prisoners of war who were captured in the beginning of 1917. Scurvy is not uncommon in Russia and her troops have felt the effect of this disease during the past four years. In some parts of the world it would seem that scurvy is still a serious problem. It is difficult to estimate, according to Hess, how common scurvy and especially infantile scurvy, is in the United States. Scurvy is a disease which requires several months to develop in man and we have no means of knowing exactly the many nutritional disorders which are in all probability latent cases

of scurvy.

Scurvy has long been recognized as a nutritional disease and practically all reports of scurvy cases have stated that its appearance was preceded by a monotonous diet which was lacking in fresh foods. When the diet was changed to one of fresh foods, especially green vegetables there was a decided improvement in the condition and recovery was brought about by continuing this diet. Orange juice and lemon juice are among the most potent antiscorbutic foods known. Lime juice, ^{which was said to have been} ~~which was~~ used on the old sailing vessels to alleviate scurvy and in Arctic expeditions, is believed by recent investigators to have been lemon juice. Chick, Hume, Skelton and Smith (1913) have found that the juice of the lime is only about one-fourth as effective as that of the lemon and that the preserved lime juice is useless. Chick and Rhodes (1913) state the juice of the raw swede (rutabaga) was found to be practically as effective as the orange juice in averting scurvy symptoms. Hess in his work in New York City with both human and experimental scurvy has pointed out the relationship of the two altho such a relation has been questioned by some of the other workers. He states that the clinical symptoms are the same but in many cases there has been a neglect of microscopic examination of the bones of the animals affected with scurvy and if this had been done some cases of animal scurvy would probably have been eliminated as rickets or pseudoscurvy. One will recall that the military posts which received a supply of potatoes had only a "taint" of scurvy. Hess and Unger (1919) have shown that potato is effective as a cure when given to ⁱnfants as well as to guinea pigs.

To Holst and Frölich (1907, 1912, 1913) we owe the discovery of experimental scurvy in animals. These investigators have shown that by feeding guinea pigs on a diet composed of cereals they were able to produce a condition which was very similar to human scurvy. The clinical pictures were very similar except that "affection of the gums is not usually found in the guinea pig." By the addition

of a fresh vegetable to the diet, such as cabbage or carrots, the animal was well nourished and showed no signs of scurvy. Jackson and Moore (1915) working with guinea pigs found that they were able to produce experimental scurvy with various diets. One series of animals was given a diet of pasturized market milk, oats, hay and water; another series was given a diet in which the pasturized milk was replaced by fresh whole milk; and still another series received a diet in which milk boiled 10 minutes was substituted for the pasturized milk. But in none of their work was the individual food intake recorded and many of the latter workers have questioned their results because of this neglect. McCollum and Pitz (1917) call attention to the peculiar anatomy of the cecum of the guinea pig which they believe^{is} conducive to constipation and state that "the undue retention of feces is the primary causes of experimental scurvy in the guinea pig." Scurvy in the guinea pig, according to these authors, is not the result of the deficiency of a specific protective substance in the food but rather due to toxic products or bacterial action. According to Pitz (1913) scurvy is due to the absorption of putrefactive products of the cecum and anything which will improve or change the intestinal flora from a putrefactive type should prevent the onset of scurvy. He found that by adding lactose to a diet of oats and milk scurvy was not only prevented but cured. Here again the amount of milk consumed was not measured and since the individual intake is so variable it is questionable whether the results obtained were as accurate as the author would lead us to believe. Hardin and Zilva (1918) used levulose, cane sugar, lactose and the uncrystallized residues from the preparation of levulose from inulin in their work with guinea pigs but none of these substances afforded any protection against scurvy. These investigators believe that the results obtained by McCollum and Pitz (1917) and Pitz (1913) was due to the enhanced consumption of fresh raw milk. Cohen and Mendel (1918) were unable to effect a cure by the use of lactose and suggest that the

results Pitz obtained may have been due to small unsuspected amounts of vitamin C which have been shown to be associated with the lactose used in certain nutrition experiments with isolated food stuffs. Drummond (1916). These same workers say that constipation is not responsible for the appearance of scurvy as McCollum and Pitz have stated, but thru its complications the severity of the disease may be enhanced. They have also found that scurvy can be produced at will by suitably chosen diets and even tho the diet contains all of the essentials of a well-balanced ration scurvy develops. These investigators have used milk in their experiments and the results which they have obtained indicate that milk does contain an antiscorbutic to a small degree.

Chick, Hume, and Skelton (1918) working at the Lister Institute have given us some very interesting results concerning the value of milk as an antiscorbutic. They have found that fresh cow's milk contains an antiscorbutic substance but in small amounts. Altho the onset of the disease was delayed and the growth of the animal was maintained in proportion to the daily consumption of milk a diet consisting almost exclusively of milk was necessary to prevent scurvy. Amounts of milk less than 50 cc. gave very little protection as death from scurvy resulted in 30 days while 50 cc. gave increased protection and the life of the animal, in one experiment, was prolonged to 75 days; when the daily intake was increased to 85-130 cc. good health was maintained thruout the period of their experiment which covered in one case a period of 113 days. The same workers have found that this antiscorbutic which is present in fresh milk is destroyed by heating in an autoclave at 120° for 1 hour, and also by drying and they state that when it is necessary to use milk in infant feeding which has been heated or dried an additional antiscorbutic substance, such as orange juice, should be added, a common pediatric practice in this country.

It seemed desirable to confirm the work of Chick, Hume, and Skelton as to

the antiscorbutic value of raw milk since there was a question concerning the work which Jackson and Moore (1916) and Pitz (1918) had done because of their failure to measure the amounts of milk actually consumed. In view of the fact that in their experiments on heated milk these English investigators had used milk autoclaved at 120° for 1 hour it was decided also to extend the study to milk subjected to somewhat less rigorous treatment. Accordingly an investigation of the antiscorbutic value of milk boiled as is customary in pediatric practice has been made. It was hoped that some experiments could be carried out upon condensed milk and milk powders, but lack of time gave no opportunity for this further study.

Before taking up the experimental work it seems advisable to describe what is understood to be experimental scurvy of the guinea pig from the clinical standpoint and also the conditions which the autopsy of a well defined case of scurvy reveals. Chick, Hume and Skelton (1918) have given the most complete statement of scurvy symptoms and the observations made in the present work have been based upon the symptoms as outlined by these authors. "The first symptoms to be observed is soreness of joints and limbs more especially of shoulders and knees, so that the animal squeaks when pressure is applied to the places. Some animals squeak when handled under any circumstances and when in perfect health; but if they are examined regularly from the beginning of the experiment it is possible to distinguish those which are feeling pain. The presence of painful members is also shown by the assumption of what we have called the 'scurvy position' which seems to indicate hemorrhage and consequent discomfort in the muscles of the limbs. The animal rests on its side and the painful leg is held off the ground and may be seen twitching. A second attitude which we have called the 'face-ache position' is also indicative of scurvy in young guinea pigs, the animal lies curled up with the side of its face pressed on the floor of the cage. This is a frequent attitude in adult guinea-pigs when in normal health but we have never

seen a young animal adopt it except when ill with scurvy. It seems to indicate hemorrhage of the jaw, with soreness and looseness of the teeth. The state of the molar teeth and of the whole gums cannot be inspected during life and it is only possible to judge of the condition by the greater or less capacity for eating and by the assumption of the 'face-ache position'." At autopsy the most noticeable condition is the hemorrhagic areas which are found subcutaneously around the shoulder and knee joints, and intramuscularly. The costo-chondral junction is enlarged and hemorrhagic and there is marked fragility of the bones. There is often a marked hemorrhage across the sternum and in some few cases there has been a noticeable loosening of the teeth.

Experimental work:

Since various workers have shown conclusively that experimental scurvy can be produced in the guinea pig on a cereal diet it was decided to make oatmeal the foundation of the diet. Oatmeal is not an adequate diet according to McCollum, Simmonds and Pitz (1917), since it is lacking in protein and in inorganic salts. It was thought that in supplementing the oatmeal with milk the protein requirements would be met by the casein, the inorganic salts would be furnished, and in addition we would have the two essentials, fat soluble A and water soluble B, which McCollum says are necessary. In the first series of experiments fresh carrots which had been boiled for 1 hour were added to the diet to give roughage since McCollum and Pitz (1917) have stated that a guinea pig can thrive only on a diet possessing such physical properties as will lead to the formation of bulky easily eliminable feces.

Series I

Six animals which had been under observation for 10 days were chosen for the first set of experiments. These ranged in weight from 312 grams to 572 grams at the beginning of the experiment. They were kept in steel cages with wire bottoms of small mesh with removable tray beneath. These cages were found much

more satisfactory for the work with milk than the cages with the metal bottoms because of the large volumes of urine which are generally excreted. Guinea pigs No. 1, 2 and 7 were given a diet of oatmeal, ad libitum, 35 grams boiled carrots and boiled milk. The milk in this work was heated to the boiling point and then removed from the flame. The daily consumption of oats was weighed and the amount of milk measured altho the animal was, in most cases, allowed to drink all it cared for. In this work attempt was made to keep the daily intake of milk up to 40 cc. or above. In order to do this it was often necessary to resort to forced feeding with a pipette. No. 1 was kept on this diet for 76 days after which the carrots were removed. On the 23rd day this animal showed clinical signs of scurvy, it continued to gain in weight altho it showed every symptom of scurvy. On the 37th day its condition seemed improved and by the 45th day there was no clinical evidence of scurvy. After removing the carrots the animal's weight began to go down (Chart I) and on the 50th day scurvy symptoms were again in evidence. It was killed on the 104th day because of a severe infection which had developed around the anus. It had been necessary to remove him from the wire cage to one with a steel bottom and because this did not drain well the animal often sat in the urine. The autopsy showed very marked subcutaneous hemorrhages around the knees and thru out the thoracic cavity. There were marked intramuscular hemorrhages in the hind legs and in the thoracic cavity. There was a very marked hemorrhage across the sternum and the costo-chondral junction was decidedly hemorrhagic. The ends of the ribs were somewhat enlarged tho there was no enlargement of the joints. The bones were very fragile and the teeth were slightly loose.

No. 2 was given the diet of oatmeal, boiled milk and boiled carrots for a period of 60 days. The carrots were then removed from the diet and on the 88th day scurvy symptoms were first noticed. The consumption of milk had been 50 cc. or above after the 20th day. Up to this time the animal had refused to cooperate

and what milk it got had to be forced down it, and even then there was much resistance. The animal's feet became very sore and it was thot best to remove it to a cage with a metal bottom. As in the case of No. 1, a serious infection resulted and the animal was killed on the 96th day. There was a rapid loss in weight after the development of the scurvy symptoms and the general condition of the animal was bad. It showed loss of appetite and evacuations were made with great difficulty. The autopsy showed marked scurvy lesions (Table I).

Guinea pig No. 7 (Table III) which was given the diet of oatmeal, boiled milk and carrots, showed clinical symptoms of scurvy on the 24th day and these continued thru out the remaining days of the experiment. It refused to eat the carrots after the 28th day. It was killed on the 71st day because it was found to be infested with lice. Autopsy revealed a well defined case of scurvy.

Guinea pig No. 6 was used as a control for this series and was given a diet of oatmeal and boiled carrots. This animal maintained itself with a slight decrease in weight (Chart II) up to the 60th day when the carrots were removed. It had shown symptoms of scurvy on the 16th day of the experiment but had recovered. There was a marked decrease in weight following the removal of the carrots and on the 64th day scurvy symptoms were quite evident. It was killed on the 84th day and the autopsy revealed the typical scurvy picture (Table III).

Guinea pigs Nos. 3 and 5 were given the same diet as Nos. 1 and 2 with the exception that the milk was raw milk. Table II shows the average daily intake of these animals for each 10-day period of the experiment. No. 3 refused to drink much milk in the beginning but later became quite greedy for it and consumed 100 cc. daily. It developed slight clinical symptoms of scurvy on the 20th day. On the 28th day the animal had a marked diarrhea which continued until the 41st day. On the 31st day the raw milk was changed to boiled milk with the hope of checking the diarrhea. The animal was very weak and sore and on the 37th day orange juice

was given with the hope of curing him. This was effective and his condition improved. Chart II, and on the 45th day the orange juice was discontinued. After discontinuing the orange juice, the animal began to fail and lost weight rapidly tho he was consuming 100 cc. of milk daily. At the beginning he was very eager for both the oatmeal and carrots but with the increased consumption of milk his appetite for these decreased. He died on the 51st day. The autopsy showed slight hemorrhages in the ribs but none in the knee joints. The death of this animal was probably due to the increased milk intake (Cohen and Mendel 1918) rather than to scurvy, since the animal was in such a weakened condition due to the severe diarrhea.

Guinea pig No. 5 lived for 27 days on the diet of oatmeal, carrots and raw milk. The milk consumption was low and on the 20th day scurvy symptoms were noticeable. The autopsy revealed a mild case of scurvy.

The boiled carrots which were given to the animals in series I evidently did not have their antiscorbutic properties destroyed as the animals which received 35 grams daily lived a longer period of time than those who received less. When milk is heated to the boiling point and then removed from the flame its antiscorbutic value is not changed appreciably. By comparing the weight curves of Nos. 1 and 2 (Chart I) with the weight curve of No. 6 (Chart II) it would seem that the addition of milk to the diet of oatmeal and boiled carrots improves the diet and the animals not only maintain themselves but grow as well.

Series II

With the next group of animals it was considered advisable to subject the milk to a more uniform treatment than had previously been done. As it was hoped that the results obtained from this work would be applicable to infant feeding the milk was heated as is customary in pediatric practice. The usual treatment in pediatric work is to bring the milk to a boil as quickly as possible and boil

for three minutes with constant stirring to prevent the formation of scum across the top. A flame was regulated so that the time required to bring the milk to the boiling point was exactly four minutes, and this was used thru out this work. The boiled carrots were not given to the animals of this group as it is believed that the carrots which are boiled for 1 hour do not have their antiscorbutic properties destroyed. This is in accord with the results obtained by Hess and Unger (1919) in which they show that 35 grams of carrots are necessary to afford protection against scurvy to guinea pig, and if the carrots are fresh boiling for 1 hour has no effect upon their potency.

The diets of the animals in this group were oatmeal and fresh raw milk in one series and in the second series milk which had been boiled as outlined above was substituted for the raw milk. Guinea pigs Nos. 10, 11, 13 and 14 were given a diet of oatmeal ad libitum and fresh raw milk. All of these animals were young ranging in weight from 139 grams to 278 grams. Table V shows the average daily intake of oatmeal and milk and the average change in weight (Chart III).

Guinea pigs Nos. 11, 13 and 14 died on the 40th, 49th and 30th days, respectively of the experiment. At the autopsy No. 11 showed marked evidence of scurvy. Clinical symptoms had been in evidence since the 32nd day. At no time during the experiment did No. 14 show any signs of scurvy and the autopsy indicated that death was due to some other cause rather than scurvy. No. 13 showed clinical symptoms of scurvy on the 34th day and at the autopsy there was evidence of a mild case of scurvy. The lungs were badly congested and the liver was not normal. Guinea pig No. 10 has been in the laboratory for 108 days and is in good health and gaining in weight. On the 31st day this animal showed clinical signs of scurvy which persisted to the 46th day (Table V). The milk intake of this animal has been gradually increased from 40 cc. to 75 cc. and it was after the intake had been increased to 30 cc. that there was a disappearance of scurvy symptoms.

Guinea pigs Nos. 8, 9, 16 and 17 were given a diet of oatmeal and boiled

milk. No. 8 died on the 33rd day and the autopsy revealed marked scurvy. The milk intake was 40 cc. thru the period of the experiment. No. 9 died on the 55th day. There was a constant decrease in weight and altho he drank milk readily he refused to eat the oatmeal. The milk intake was increased but this resulted in a diarrhea, and it is believed that the amount of milk which he consumed was not sufficient to maintain life. At one time, 38th day, the knee joints were slightly sore but the autopsy did not reveal scurvy.

Guinea pig No. 16 was killed at the end of the 37th day because of weakened condition. The autopsy gave no evidence of scurvy. The milk intake of this animal had been 40cc. or more thru out the experiment.

Guinea pig No. 17 has been in the laboratory for 60 days. It shows clinical symptoms of scurvy and its general condition is bad altho the animal is not decreasing in weight very rapidly.

When the guinea pig receives a diet of oatmeal supplemented with milk, either raw or boiled, the life of the animal is prolonged in nearly all cases. In comparing the results of this series with the results of series I there is not the difference in the effect of the boiled milk as one might expect. The animal must receive an average daily intake of more than 40 cc. of milk in order to have any marked effect in preventing the onset of scurvy. (Nos. 8, 16 and 11) No. 10 has continued to gain in weight steadily altho the clinical symptoms of scurvy were very evident. The boiled carrots which were fed in series I prolonged the life of the animals for a longer period of time than does milk alone unless taken in large quantities.

CONCLUSIONS

Altho it has been impossible to make a complete study of this problem the following conclusions have been made from the experimental data.

1. Carrots which are boiled one hour do not have their antiscorbutic value



entirely destroyed.

2. The consumption of large quantities of milk by guinea pigs may cause marked intestinal disturbances resulting in severe diarrhea.

3. There is a decided variation among the animals in the consumption of milk and the results which Jackson and Moore (1915) and Pitz (1913) have obtained are probably due to their failure to recognize this fact.

4. Raw milk has an antiscorbutic property which is present to a small degree and which is destroyed somewhat by heating as is customary in pediatric practice.

5. Boiled milk, as well as raw milk, does improve the diet of oatmeal and the life of the guinea pig is prolonged and the appearance of scurvy symptoms delayed when an intake of 40 cc. or more of milk is consumed.

6. Guinea pigs may increase in weight very markedly while showing mild clinical scurvy. The development of ^{severe} scurvy clinically is almost invariably accompanied by marked loss in weight.

7. From this work it seems advisable to recommend that an antiscorbutic food should be included in the diet of infants who are given milk which has been subjected to heating.

I wish to acknowledge my indebtedness to Dr. Howard L. Lewis, under whose supervision this work has been done, and whose frequent suggestions have made possible the completion of the work.

TABLE I
Average Daily Weight and Intake of Food

Guinea Pig No. 1							Guinea Pig No. 2						
Food Intake							Food Intake						
Period	Days	Weight gms.	Oats gms.	Milk cc.	Carrots gms.	Remarks	Period	Days	Weight gms.	Oats gms.	Milk cc.	Carrots gms.	Remarks
I	1-10	320	8.1	33	35	Milk, boiled	I	1-10	450.6	13.2	24.3	35	Milk, boiled
II	10-20	337	10.3	31	35		II	10-20	464.6	19.5	38.4	35	
III	20-30	333	12.0	35	35	Scurvy signs on 23 day	III	20-30	498.0	16.5	59.3	35	
IV	30-40	365	13.0	38	35		IV	30-40	587.4	17.9	76.8	35	
V	40-50	393	13.0	39	35	No scurvy signs on 45 day	V	40-50	655.6	14.8	67.0	35	
VI	50-60	422	13.0	43	35		VI	50-60	672.6	18.2	72.5		Carrots removed on 60 day
VII	60-70	458	13.0	64	35		VII	60-70	694.6	19.7	66.5		
VIII	70-80	499	14.0	66	35	Carrots removed on 76 day	VIII	70-80	694.0	15.2	72.1		
IX	80-90	481	13.0	60	0	Slight scurvy signs on 90 day	IX	80-90	662.2	13.2	59.5		Scurvy signs on 83 day
X	90-100	425	8.0	39	0	96 day - marked symptoms	X	90-96	559.5	3.5	28.3		Killed on 96 day
XI	100-104	369	5.0	45	0	Killed on 104 day Scurvy							Marked scurvy

TABLE II

Average Daily Weight and Intake of Food

Guinea Pig No. 3										Guinea Pig No. 5									
Period					Food Intake					Food Intake					Remarks				
Days					Oats					Oats					Carrots				
gms.					gms.					gms.					gms.				
cc.					cc.					cc.					cc.				
gms.					gms.					gms.					gms.				
cc.					cc.					cc.					cc.				
gms.					gms.					gms.					gms.				
cc.					cc.					cc.					cc.				
gms.					gms.					gms.					gms.				
cc.					cc.					cc.					cc.				
gms.					gms.					gms.					gms.				
cc.					cc.					cc.					cc.				
gms.					gms.					gms.					gms.				
cc.					cc.					cc.					cc.				
gms.					gms.					gms.					gms.				
cc.					cc.					cc.					cc.				
gms.					gms.					gms.					gms.				
cc.					cc.					cc.					cc.				
gms.					gms.					gms.					gms.				
cc.					cc.					cc.					cc.				
gms.					gms.					gms.					gms.				
cc.					cc.					cc.					cc.				
gms.					gms.					gms.					gms.				
cc.					cc.					cc.					cc.				
gms.					gms.					gms.					gms.				
cc.					cc.					cc.					cc.				
gms.					gms.					gms.					gms.				
cc.					cc.					cc.					cc.				
gms.					gms.					gms.					gms.				
cc.					cc.					cc.					cc.				
gms.					gms.					gms.					gms.				
cc.					cc.					cc.					cc.				
gms.					gms.					gms.					gms.				
cc.					cc.					cc.					cc.				
gms.					gms.					gms.					gms.				
cc.					cc.					cc.					cc.				
gms.					gms.					gms.					gms.				
cc.					cc.					cc.					cc.				
gms.					gms.					gms.					gms.				
cc.					cc.					cc.					cc.				
gms.					gms.					gms.					gms.				
cc.					cc.					cc.					cc.				
gms.					gms.					gms.					gms.				
cc.					cc.					cc.					cc.				
gms.					gms.					gms.					gms.				
cc.					cc.					cc.					cc.				
gms.					gms.					gms.					gms.				
cc.					cc.					cc.					cc.				
gms.					gms.					gms.					gms.				
cc.					cc.					cc.					cc.				
gms.					gms.					gms.					gms.				
cc.					cc.					cc.					cc.				
gms.					gms.					gms.					gms.				
cc.					cc.					cc.					cc.				
gms.					gms.					gms.					gms.				
cc.					cc.					cc.					cc.				
gms.					gms.					gms.					gms.				
cc.					cc.					cc.					cc.				
gms.					gms.					gms.					gms.				
cc.					cc.					cc.					cc.				
gms.					gms.					gms.					gms.				
cc.					cc.					cc.					cc.				
gms.					gms.					gms.					gms.				
cc.					cc.					cc.					cc.				
gms.					gms.					gms.					gms.				
cc.					cc.					cc.					cc.				
gms.					gms.					gms.					gms.				
cc.					cc.					cc.					cc.				
gms.					gms.					gms.					gms.				
cc.					cc.					cc.					cc.				
gms.					gms.					gms.					gms.				
cc.					cc.					cc.					cc.				
gms.					gms.					gms.					gms.				
cc.					cc.					cc.					cc.				
gms.					gms.					gms.					gms.				
cc.					cc.					cc.					cc.				
gms.					gms.					gms.					gms.				
cc.					cc.					cc.					cc.				
gms.					gms.					gms.					gms.				
cc.					cc.					cc.					cc.				
gms.					gms.					gms.					gms.				
cc.					cc.					cc.					cc.				
gms.					gms.					gms.					gms.				
cc.					cc.					cc.					cc.				
gms.					gms.					gms.					gms.				
cc.					cc.					cc.					cc.				
gms.					gms.					gms.					gms.				
cc.					cc.					cc.					cc.				
gms.					gms.					gms.					gms.				
cc.					cc.					cc.					cc.				
gms.					gms.					gms.					gms.				
cc.					cc.					cc.					cc.				
gms.					gms.					gms.					gms.				
cc.					cc.					cc.					cc.				
gms.					gms.					gms.					gms.				
cc.					cc.					cc.					cc.				
gms.					gms.					gms.					gms.				
cc.					cc.					cc.					cc.				
gms.					gms.					gms.					gms.				
cc.					cc.					cc.					cc.				
gms.					gms.					gms.					gms.				
cc.					cc.					cc.					cc.				
gms.					gms.					gms.					gms.				
cc.					cc.					cc.					cc.				
gms.					gms.					gms.					gms.				
cc.					cc.					cc.					cc.				
gms.					gms.					gms.					gms.				
cc.					cc.					cc.					cc.				
gms.					gms.					gms.					gms.				
cc.					cc.					cc.					cc.				
gms.					gms.					gms.					gms.				
cc.					cc.					cc.					cc.				
gms.					gms.					gms.					gms.				
cc.					cc.					cc.					cc.				
gms.					gms.					gms.					gms.				
cc.					cc.					cc.					cc.				
gms.					gms.					gms.					gms.				
cc.					cc.					cc.					cc.				
gms.					gms.					gms.					gms.				
cc.					cc.					cc.					cc.				

TABLE III

Average Daily Weight and Intake of Food

Guinea Pig No. 6							Guinea Pig No. 7						
Food Intake							Food Intake						
Period	Days	Weight gms.	Oats gms.	Milk cc.	Carrots gms.	Remarks	Period	Days	Weight gms.	Oats gms.	Milk cc.	Carrots gms.	Remarks
I	1-10	484	14	--	35		I	1-10	395	13	30	30	Milk, boiled
II	10-20	500	17	--	35		II	10-20	367	9	30	30	
III	20-30	498	19	--	35		III	20-30	343	6	44	23	Scurvy symptoms 24 day 28 day
IV	30-40	492	16	--	35		IV	30-40	345	10	33	--	refused to eat carrots
V	40-50	473	12	--	35		V	40-50	297	7	29	--	
VI	50-60	461	14	--	35	Carrots removed on 60th day	VI	50-60	306	7	36	--	
VII	60-70	417	10	35	--	Raw milk added on 68th day	VII	60-70	314	7	47	--	Killed 71st day Scurvy
VIII	70-80	336	10	52.1	--								
IX	80-84	360	6	40	--	Killed 84th day Scurvy							

TABLE IV

Average Daily Weight and Intake of Food

Guinea Pig No. 8						Guinea Pig No. 9					
Period	Days	Food Intake			Remarks	Period	Days	Food Intake			Remarks
		Weight gms.	Oats gms.	Milk cc.				Weight gms.	Oats gms.	Milk cc.	
I	1-10	213	7	40	Milk, boiled	I	1-10	275	1.4	45	Milk, boiled
II	10-20	224	9	40		II	10-20	235	.4	50	
III	20-30	199	7	40		III	20-30	206	3.2	50	
IV	30-33	155	2	40	Dead 33rd day Scurvy	IV	30-40	214	3.1	54	
						V	40-50	184	1.5	60	
						VI	50-55	161	0	44	Dead 55th day No scurvy

Guinea Pig No. 16						Guinea Pig No. 17					
Period	Days	Food Intake			Remarks	Period	Days	Food Intake			Remarks
		Weight gms.	Oats gms.	Milk cc.				Weight gms.	Oats gms.	Milk cc.	
I	1-10	311	6	40	Milk, boiled	I	1-10	344	7	42	Milk, boiled
II	10-20	287	9	47		II	10-20	344	6	36	
III	20-30	262	12	48		III	20-30	317	6	52	
IV	30-37	234	6	55	filled at end of 37th day no scurvy symptoms	IV	30-40	314	12	53	
						V	40-50	305	8	59	45th day ankles sore

TABLE V

Average Daily Weight and Intake of Food

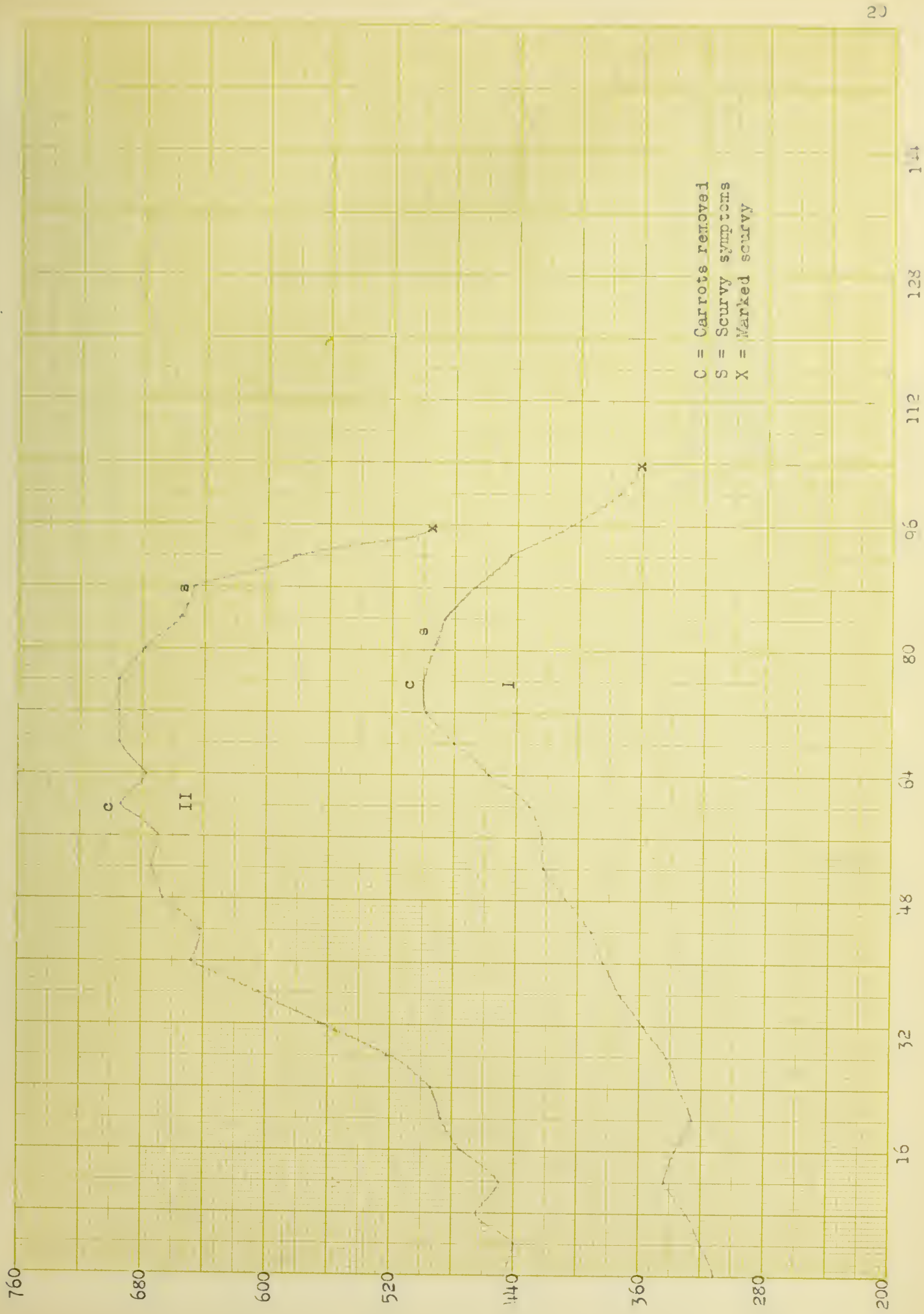
Guinea Pig No. 10						Guinea Pig No. 11					
Period	Days	Weight gms.	Food Intake		Remarks	Period	Days	Weight gms.	Food Intake		Remarks
			Oats gms.	Milk cc.					Oats gms.	Milk cc.	
I	1-10	138	8	13	Milk, raw	I	1-10	184	5	34	Milk, raw
II	10-20	198	6	38		II	10-20	196	6	45	
III	20-30	216	8	40		III	20-30	217	6	43	
IV	30-40	237	8	40	34th day scurvy signs	IV	30-40	201	5	34	32nd day scurvy symptoms
V	40-50	254	7	40	47th day no scurvy						31st day marked scurvy
VI	50-60	285	8	50							
VII	60-70	289	10	50							
VIII	70-80	313	10	50							
IX	80-90	332	5	75							
X	90-100	367	8	75							
XI	100-110	385	9	75	Still lives						

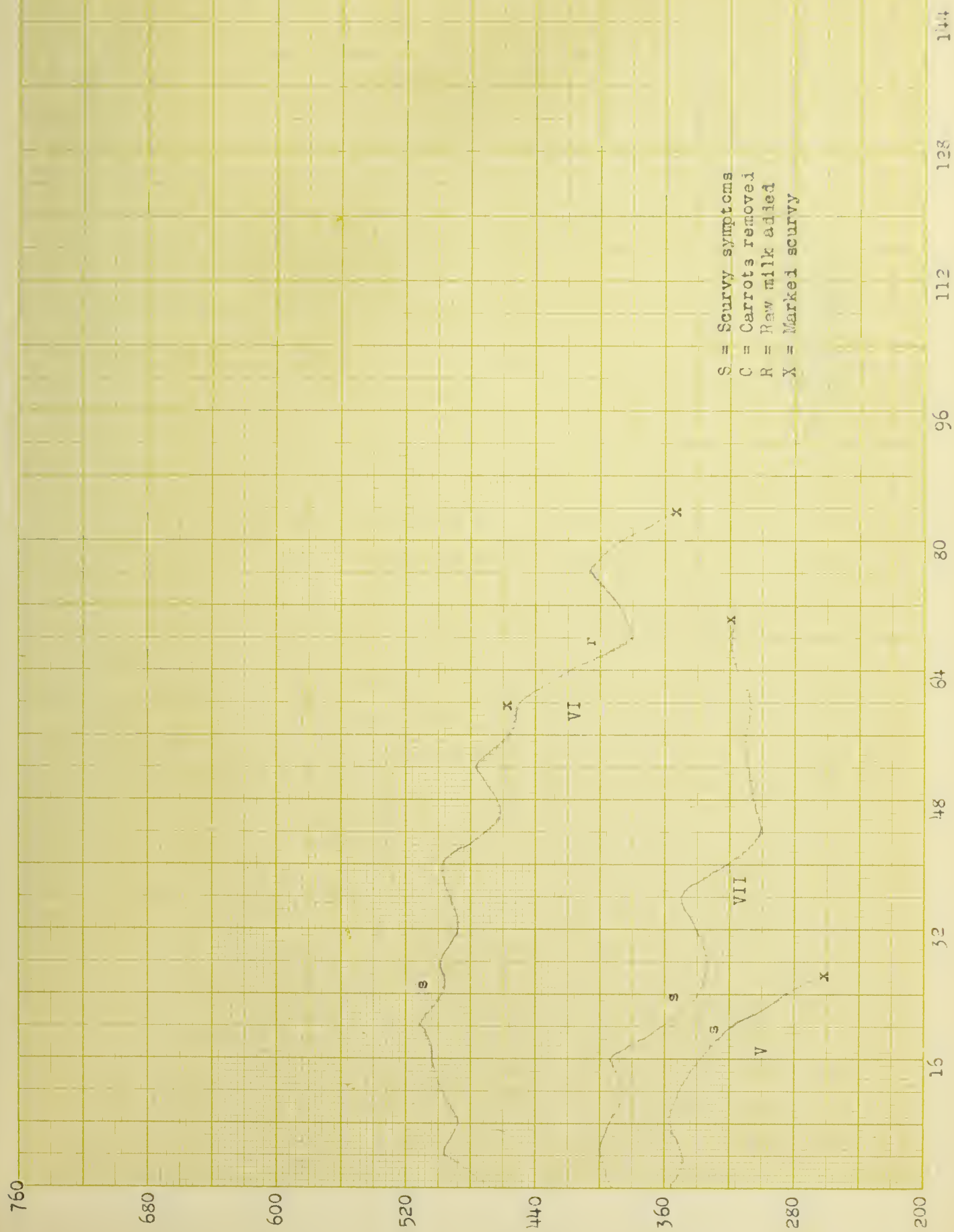
TABLE V (CONTINUED)

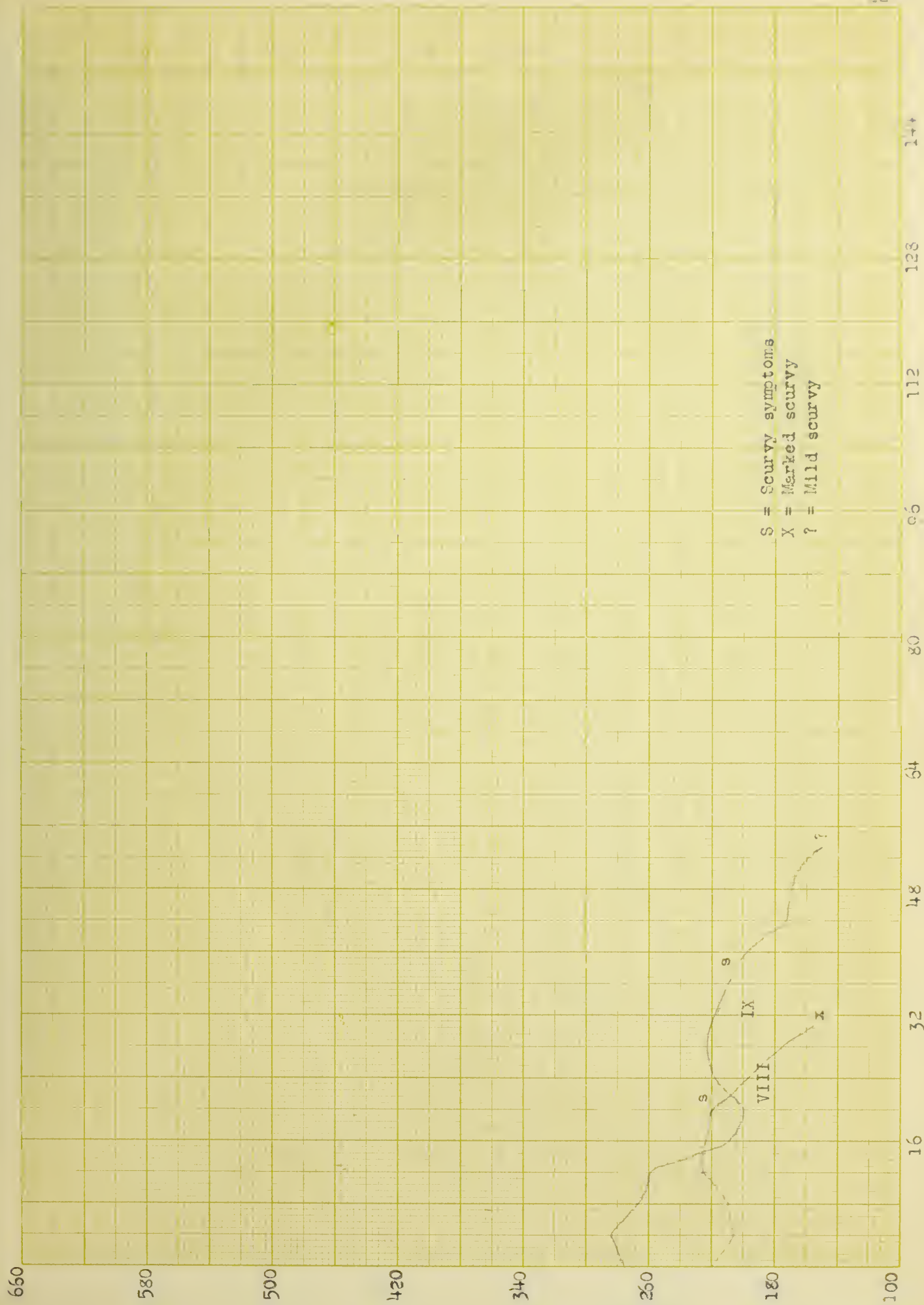
Average Daily Weight and Intake of Food											
Guinea Pig No. 13						Guinea Pig No. 14					
Period	Days	Weight gms.	Food Intake		Remarks	Period	Days	Weight gms.	Food Intake		Remarks
			Oats gms.	Milk cc.					Oats gms.	Milk cc.	
I	1-10	230	8	39	Milk, raw	I	1-10	270	5	41	Milk, raw
II	10-20	257	4	39		II	10-20	261	4	36	
III	20-30	243	8	47		III	20-30	253	5	48	40th day very weak. Killed No scurvy.
IV	30-40	252	6	42							
V	40-50	217	5	33	44th day slight scurvy symptoms Dead 50th day Enlargement of costo-chondral junction. Lungs congested. Mild scurvy. Pneumonia						

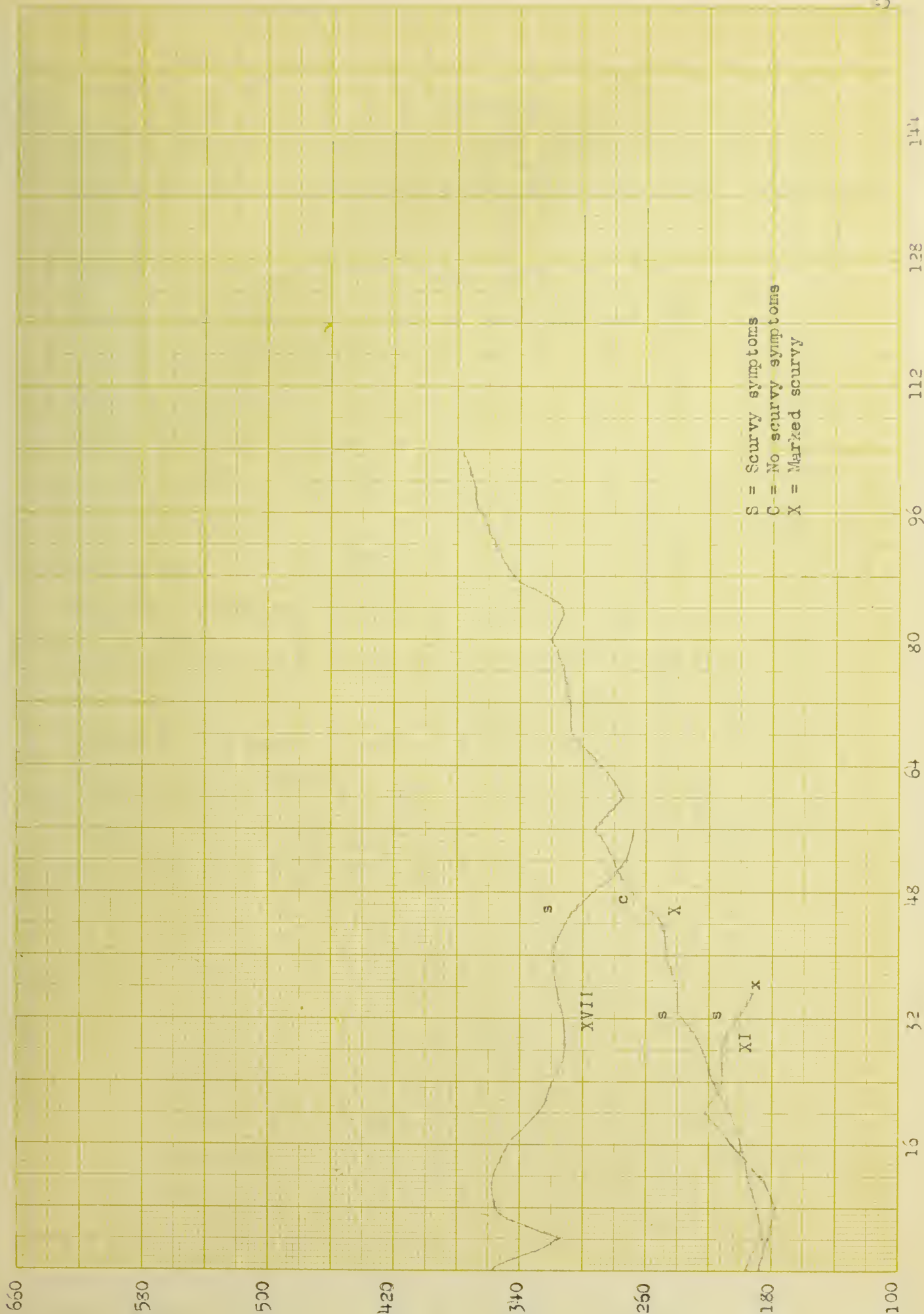
TABLE VI

Guinea pig No.	Initial	Weights Maxi- mum.	Final	Dura- tion	Diagnosis	Remarks
Oatmeal, boiled milk and boiled carrots						
	gms.	gms.	gms.	days		
1	312	500	361	104	Scurvy	
2	447	706	492	96	Scurvy	
7	395	401	319	71	Scurvy	
Oatmeal, raw milk and boiled carrots						
3	572	602	402	51	Mild scurvy	Severe diarrhea caused weakened condition.
5	354	357	260	27	Mild scurvy	
6	465	512	350	84	Scurvy	
Oatmeal and boiled milk						
8	220	227	147	33	Scurvy	
9	276	283	150	55	No scurvy	
16	309	344	215	37	" "	
17	355	374	258	60+	Scurvy symptoms.	Animal still alive tho shows clinical scurvy.
Oatmeal and raw milk						
10	196	404	404	114+	Normal	Animal still alive. Seemingly has fully recovered from scurvy symptoms noticable on 34th day.
11	189	223	209	39	Scurvy	
13	278	232	203	44	Scurvy (?)	Pneumonia was the real cause of death tho some scurvy lesions visible.
14	277	302	250	40	No scurvy	
						Killed because of weakened condition.





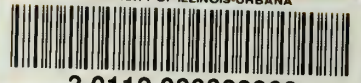




BIBLIOGRAPHY

- Chick, H., Hume, E. M., Skelton, R. F., 1918. Biochem. Jour., xii, 131.
- Chick, H., Hume, E. M., Skelton, R. F., and Smith, A. L., 1918. Lancet, cxcv, 735.
- Chick, H., and Rhodes, H., 1918. Lancet, cxcv, 774.
- Cohen, F., and Wendel, L. B., 1918. J. Biol. Chem. xxxv, 425.
- Drummond, J. C., 1916. Biochem. J., x, 89.
- Hardin, A., and Zilva, S. S., 1918. Biochem. J., xii, 270.
- Harvier, P., 1917. Paris med. vii, 394.
- Hess, A. F., 1913. J. Am. Med. Assoc., 71, 941.
- Hess, A. F., and Unger, L. J., 1919. ^{Am.} J. of Dis. of Children, xvii, 221.
- Hess and Unger, 1919. ^{Soc.} Proc. ^{Exp.} Biol. and Med., xvi, 52.
- Holst, A., and Frölich, T., 1907. J. Hygiene, vii, 63+.
- Holst and Frölich, 1912. Z. Hyg. u. Infektionskrankh., lxxii, 1.
- Holst and Frölich, 1913. Z. Hyg. u. Infektionskrankh., lxxv, 334.
- Jackson, L., and Moore, 1916. J. Inf. Dis. xix, 478.
- McCollum, E. V., and Pitz, W., 1917. J. Biol. Chem. xxxi, 229.
- McCollum, E. V., Simmonds, N., and Pitz, W., 1917. J. Biol. Chem. xxix, 341.
- Pitz, W., 1918. J. Biol. Chem., xxxiii, 471.

UNIVERSITY OF ILLINOIS-URBANA



3 0112 086833263